as an intramuscular injection, but which left a fine trail of mercurial stomatites behind it. One man was using the sosoiodolate of mercury.

During my journey I heard much of atoxyl as a remedy for syphilis, but saw very little of it used. One man said he used it if he found mercury to disagree. After using atoxyl for a time he would drop it, and recur to the use of mercury, which he would now expect to agree. To get decided antisyphilitic effects from atoxyl, however, it has to be pushed to its physiologic limit, and there is danger that the patient may become temporarily blind. There is no occasion for insisting on the gravity of such a situation, as a perambulating case immediately becomes a hospital case. It is no wonder that many are entirely opposed to employing this drug against syphilis. Max Joseph, for instance, not alone did not advise atoxyl as an antiluetic remedy, but warned his hearers most emphatically against its use.

Our return home was as uneventful and pleasant as any part of our journey. It is profitable and enjovable to see other cities, other peoples, other ways of living, but this is only a foretaste to the pleasure of again seeing San Francisco's magnificent bay.

"Happy indeed is he who returns home after a good voyage."

SUBJECTIVE SYMPTOMS AND PAINFUL SENSATIONS IN HEART DISEASE.*

By E. SCHMOLL, M. D., San Francisco.

The subjective symptoms and painful sensations in heart disease, and the reflex symptoms due to disturbed function of the heart, have attracted very little attention except in angina pectoris. In this disease, pain has monopolized the attention of observers to the exclusion of other symptoms.

It has been held an axiom that heart disease runs a painless course as long as the heart is well compensated. Potain used to say that when a patient complained of pain about the heart the presumption was against his having any heart lesion.

Close study of a great number of diseased hearts during the last two years has led me to the conviction that in every case of heart disease we have painful sensations, and that these sensations range from the hardly perceptible sense of constriction in mitral insufficiency, to the annihilating pain of coronary sclerosis.

The study of the symptoms during an attack of angina pectoris and the symptoms occurring between attacks, and the checking up of these observations in ordinary cases of heart disease, have shown me that the symptoms are due to a segmental lesion of the spinal cord caused by reflex irritation from the heart. This reflex irritation takes place in every case of heart disease. The intensity of the symptoms depends on many factors, but, in a general way, is proportionate to the intensity of the pathological lesion.

In the heart disease of most unfavorable prognostication—coronary sclerosis—the symptoms are most severe.

Reflex symptoms of heart disease can be divided into three groups: sensory, motor and vaso-motor symptoms.

At certain times, these symptoms exacerbate for a short period and are felt as attacks. The same symptoms, however, are present more or less constantly all the time.

We shall begin a discussion of the reflex phenomena of heart diseases with an analysis of the symptoms present in a painful attack caused by coronary sclerosis.

Our attention was first called to these symptoms by Mackenzie's brilliant researches on referred pain in visceral lesions, and confirmed and put on a more solid basis by the work of Head. The application of these researches to an explanation of the symptoms of angina pectoris was first dwelt upon by Gibson, in his well-known work on neuroses of the heart.

(a) The sensory symptoms can be divided into sharply defined groups: firstly, the sensation of impending death; and, secondly, pain in the heart and surrounding structures. While the sensation of impending death monopolizes the patient's attention and stands in the foreground of his description, our interest is principally concentrated on the pain and its irradiations. In the great majority of cases pain is retrosternal, and thence invades the eighth cervical and first dorsal segment. Many segments may be involved, very frequently the fifth and sixth cervical, occasionally the lower dorsal segments. I have found that the pain may irradiate in practically every segment, between the second cervical and the twelfth dorsal. Usually, several zones of pain exist, between which pain free areas are interspersed. In the majority of cases, the pain begins in the middle line and radiates toward the periphery. In other cases, pain appears over the whole area supplied by the corresponding segment at once.

In a few cases, I have seen the pain start from the periphery and progress toward the middle line (beginning of pain in the fingers). In most cases the pain is left-sided. In such event the lesion corresponds to left-sided affection—either a disease of the aortic valves, of the aorta itself, or of the coronary arteries.

Sometimes the pain is bi-lateral. Iu such cases, I have always been able to find a right heart lesion. In these infrequent right-sided cases, the mitral valve is usually affected. In a case observed lately, the patient suffering from mitral insufficiency with a dilated right ventricle, the pain started in the precordial region and radiated to the right side within the boundaries of the third and fourth dorsal segments. Pain existed in the eighth cervical and first dorsal segment of the right side, radiating downward into the fingers.

(b) Motor symptoms can show themselves either in the form of irritation or paralysis. As

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an irritative symptom, we consider the sense of constriction around the chest, which the patient usually compares to the constriction of a band, or to an iron hand grasping the heart. This sensation is caused by a tonic contraction of the intercostal muscles.

Very frequently we find tonic contraction of the pectoralis major, corresponding to the spasm of the abdominal muscles over the inflammed peritoneum—a point which Mackenzie first drew attention to. In other cases, I have observed clonic contractions. In the case of right-side angina pectoris mentioned above, clonic contractions of the pectoralis major, deltoid muscle, and the muscles of the forearm occurred with every attack of pain. In such cases, as the pain passes off, very frequently paralytic symptoms follow the symptoms of irritation. In the great majority of cases, however, the paralytic symptoms are pronounced throughout the attack, without any preceding irritative symptoms. The left arm is usually powerless and cannot be elevated. The patient is unable to hold things in the left hand. Sometimes the motor symptoms are more pronounced than the sensory. I have seen a case in which typical attacks of angina pectoris alternated with attacks of transitory paresis of the left arm.

I treated a merchant, age fifty-four years, in whom, beside the signs of general arterio-sclerosis, a dilatation of the arch of the aorta was found. Several attacks of typical angina occurred after unusual efforts or mental strain. During these attacks, the patient observed that he was unable to elevate his left arm. Occasionally attacks occurred, caused by the same factors, in which the only symptom present was paresis of the left arm without any pain whatsoever. These attacks lasted but a few minutes, but they caused mental anguish not less than that which was caused in the same patient by the painful attack of angina pectoris.

(c) Vaso-motor symptoms. Changes in the blood distribution during an attack have never been missing in any attack I have seen, nor in any description of an attack given me by an intelligent patient. Usually, the vaso constriction can be first observed in the segment into which the pain irradiates, and only later it attacks the blood supply of the rest of the skin.

I have observed two cases in which the first sign of a beginning attack consisted of such a vaso constriction of the left hand that it resembled the hand of a corpse. In other cases, vaso dilatation precedes vaso constriction, the hand becoming cyanotic.

In some cases, vaso-motor symptoms are the most marked and occur with very little sensory or motor disturbance. Such cases were first described by Nothnagel as vaso-motor angina. Curshman has recently published the records of two such cases, and has proven by autopsy that they were caused by coronary sclerosis.

Examination of the patient at intervals between attacks shows that the same symptoms exist in an attenuated form, and that sensory, motor and vaso-motor disturbances are produced in the segments affected during the attack.

(a) Sensory symptoms exist in the form of hyperaesthesia of the segments, in which the pain prevails during the attack. This hyperaesthesia can be shown by any method used for ordinary neurological examinations. Usually, I follow the sternum with a pin, which I apply with equal pressure over the whole surface. As soon as the hyperaesthetic area is touched, the patient complains of being pricked by the pin, or of a burning sensation.

These hyperaesthetic areas, to which Mackenzie and Head first drew attention, are absolutely constant. I have never missed them in any case of angina pectoris. Their objective character is proven, as Mackenzie has first shown, by the appearance of a goose-skin reflex over the area of the diseased segment, and by dilatation of the pupils.

Hyperaesthesia involves not only the skin, but also the underlying muscles, as is easily shown by squeezing of the muscles corresponding to the involved segments. This hyperaesthesia corresponds, as already stated, to the distribution of the spinal segments, and to the distribution and irradiation of the pain during the attack. A very conclusive demonstration of this I saw lately in the case of right-sided angina before mentioned. In this case, beside the irradiation in the eighth cervical and first dorsal segment, pain occurred in the right lumbar region and the hip joint. Examination showed typical hyperaesthesia over the eighth cervicle and first dorsal segment, and the second zone over the third and fourth dorsal segments. Corresponding to the third zone of pain, typical hyperaesthesia could be shown in the first and second lumbar segments.

In the intervals between attacks, patients are by no means free from pain, but complain more or less constantly of painful sensations over the hyperaesthetic areas. There is usually some shooting pain, or distinct soreness over the afflicted parts.

Sometimes the pain feels like a rheumatic pain, thus accounting for the painful sensations in the left shoulder-joint, which heart patients often complain of. Very frequently we find shooting pain in the left arm most intense around the elbow. In other cases, the patient complains more of paraesthesias of numbness in the last two fingers. In other cases, we find, instead of a hyperaesthesia, an anasthesia, in segmental distribution. In a case described by Gibson, anaesthesia of the left side of the thorax and the inside of the left arm were found.

(b) The motor symptoms in the interval between attacks consist of paretic conditions, or irritative symptoms.

Generally, the tonus of the muscles corresponding to the hyperaesthetic areas is increased. This is most easily detected in the pectoral muscle, which, in comparison with the muscle of the other side, is decidedly hypertonic. This hypertonicity, however, does not correspond to an increase in strength. A comparison with the corresponding muscles of the

right side shows that strength is diminished in very much greater degree than would correspond to the physiological difference.

Very frequently the left arm measures 1½ to 2½ centimeters less than the right arm.

A typical case of paralysis following repeated attacks of angina pectoris, in which the patient lost the ability to grasp anything, or to carry an object in his hands, has been described by Gibson. Mechanical and electrical irritability was considerably increased, while the muscles themselves presented considerable atrophy.

Complete paralysis of the muscles of the diseased segments is rare, and I have never observed it. However, several cases of this kind have been described. In a case reported by Eichhorst, paralysis, with reaction of degeneration, had occurred in the muscles innervated by the ulnar nerve in a patient presenting the symptoms of angina pectoris.

(c) Vaso-motor symptoms, in the intervals between attacks, are usually not as well marked as the motor and sensory symptoms. While the action of the vaso-constrictors is usually more pronounced in the attack, vaso-dilatation prevails in the interval between attacks, so that the hands usually present a cyanotic tinge due to vaso-dilatation. In some cases, this condition alternates with attacks of vaso-constriction, in which other symptoms of angina are missing. In these, such a vaso-constriction takes place that the hand becomes absolutely exsanguinated, and the symptoms resemble those of Raynaud's disease to such a degree that the differential diagnosis is often a matter of great difficulty.

I have observed a number of these cases, and shall report them in the near future. The report of one case, referred to me by Dr. Philip Thomas, will be sufficient to show the principal points. The patient, a female, age twenty-nine years, presents the typical symptoms of aortic insufficiency, complicated with typical attacks of anginoid character, in which, beside the pain, paresis of the left arm and complete anemia of the finger tips are very pronounced. Occasionally, attacks occur in which the blood circulation through the fingers seems to be entirely stopped, and the hands resemble those of a corpse. These attacks last for several hours, and are not accompanied by any appreciable amount of pain. They are very frequently caused by exterior influences, as immersion of the hands in cold water. Evidently the explanation of this phenomena can be found in an increased excitability of the reflex-arch.

As I have already stated, similar symptoms affecting the motor, sensory, and vaso-motor functions of certain spinal segments more or less identical with the symptoms of angina pectoris occur in heart diseases without coronary sclerosis. Attacks may be observed in which patients complain of pain over the chest, in the left arm, paresis of the left arm and disturbances in vaso-motor regulation.

These symptoms occur in cases in which the left ventricle or the aorta is diseased. They are especially pronounced in cases of insufficiency of the aortic valves and dilatation of the arch of the aorta. Very frequently they are met with in cases of post-infectious myocarditis, and show themselves under these conditions principally in paretic conditions of the left arm, occurring after fatigue.

A description of the symptoms would be only a repetition of the above-described phenomena. A better insight into the conditions alluded to may be gained by the study of the history of the following cases: A typical example of anginoid pain, caused by fatty heart, may be found in the following history: Mrs. B., thirty-four years old; very obese, complains of shortness of breath and great weakness after slight exertion. Her heart is slightly enlarged to the right. Its tones sound very distant. Over the apex the first sound is prolonged, otherwise no signs of insufficiency of the heart can be demonstrated. She suffers from attacks caused by exertion or excitement or by overeating. They consist of more or less distressing pain over the region of the praecordium, radiating to the neck and to the left arm. The pain causes a sense of constriction complicated by a sensation of fear. At the same time she feels as if somebody grasped her neck and choked her. Palpitations occur in some attacks, but they are by no means a constant symptom. During attacks the inside of the left arm becomes anaesthetic and the fingers feel very numb. After the attack has lasted for some time, the arm becomes very weak so that she cannot perform any work with it. Duration of the attacks varies from a few hours to one or two days.

On examination hyperaesthetic areas are found, one corresponding to the third and fourth cervical segment, and a second one to the eighth cervical and first dorsal. Symptoms of motor irritation in the first zone explain the very pronounced sensation of choking. The objectivity of the hyperaesthesia is demonstrated by the occurrence of the goose-skin reflex and the dilatation of the pupils.

Under appropriate treatment, these symptoms, and with them the hyperaesthesia, disappeared.

The following case illustrates the anginoid symptoms in mitral stenosis: The patient, twenty-one years old, complained for the first time of heart symptoms about two years ago, after an attack of pleurisy. Dyspnoea on lying down and palpitations on exertion were complained of. There is a constant sense of pressure over the heart like a weight compressing the thorax. At times the pain becomes sharper, and shoots down the left arm into the third and fourth fingers, while in the chest there is a sensation of a hand grasping the heart. Sometimes the left arm becomes paralyzed during such an attack, and the patient is unable to perform any work with her left hand. These attacks come most frequently after some undue effort, especially if patient has been running upstairs. Sometimes the paralysis comes on without pain. Here, too, the relation of excessive muscular work to the attack is apparent. The attack always begins with cyanosis of both hands. On examination the signs

of a double mitral affection are found. There is typical hyperaesthesia over the third and fourth dorsal and the first dorsal and eighth cervical segment.

Similar symptoms I have seen in a great many affections of the heart, especially in the cases of aortic disease. In all these, the typical hyperaesthesias could be made out. The subjective symptoms and the irradiation of the pain corresponded to the extension of hyperaesthesia.

The pathogenesis of these symptoms found in heart disease extending over certain spinal segments and affecting the motor, sensory and vasomotor areas supplied by these nervous centers, has already been discussed, as far as the sensory symptoms go, by Mackenzie and Head. Mackenzie's conception of the phenomenon seems to me to explain best the symptoms discussed in this paper. He thinks that by the disease of the heart a constant stimulus irritates the nervous system of the heart—the sympathetic nerve. This constant irritation of the sympathetic nerve leads to an irritation of the spinal segment, at which the heart fibres connect with the spinal cord. The irritation of the sensory part of the spine leads to a sensation, which, according to the law of Muller, is projected into the periphery supplied by the nerves of the spinal segment: the irritation of the sensory part may become so strong that the cells get overtired, and are incapable of conducting the stimulus-producing anaesthesia. Similar conditions prevail for the motor part of the spinal chord. Irritation of the spinal segment leads to tonic or clonic contraction of the muscles, receiving their innervation from this spinal segment. Long-lasting irritation may lead to paralysis. Similar conditions prevail for the vasomotor disturbances. The innervation of the vasomotor processes has been definitely shown to be of the same segmental character as motor or sensory innervation.

According to our conception of the symptoms of angina pectoris, these originate in every case of heart affection, especially if the left ventricle is diseased. They are caused by the constant irritation of the corresponding spinal segments, through the sympathetic nervous system innervating the heart.

Differentiation of the symptoms of angina pectoris due to coronary sclerosis, and those occurring in other pathological conditions of the heart, is, at times, very difficult, as there exists only a difference in intensity, not in character. The lesion interfering most with the lesion of the heart, coronary sclerosis, is apt to lead to the most accentuated symptoms—to the classical attack of angina pectoris.

Prolonged observation of the patient in regard to the causative factor of the attacks, their duration, intensity of the pain, etc., will, in the great majority of cases, permit a differentiation and allow us to give a correct prognosis.

COOPER COLLEGE SCIENCE CLUB.

Dr. Schmoll, presenting case of infectious meningomyelitis:

The case which I have the honor to demonstrate tonight has been a very interesting one to me on account of a very unusual spinal lesion presenting considerable difficulties in diagnosis. The patient is thirty-two years old. There is nothing in his family or past history which would shed any light on the present condition except that he had a chancre about ten years ago, but without any secondary symptoms. I may mention here that examination does not show any symptoms of syphilis, so that it remains doubtful whether the patient ever had syphilitic infection or not. The present sickness began about three months ago with pain in his back, getting worse when he walked downstairs, and especially if he made a misstep. This pain would occur no matter on which leg he would step. At the time he was a patient in our clinic at Cooper College, and as no objective sign could be found, his case was diagnosed as one of muscular rheumatism. Slowly the pain progressed, extending from the right lumbar region across his abdomen and then crossing the middle line, finally encircling the whole body at the height of the umbilicus. About five or six weeks ago patient began to notice that he had a numbness in his left leg extending over the region of the first and second lumbar segments, and over the anterior aspect of his leg down to the knee. About three or four weeks ago numbness was noticed in his right leg and finally after another week patient began to notice his legs getting weaker, and a few days ago he was completely paralyzed. At the same time paralysis of the bladder and rectum occurred, patient being unable either to void urine or pass feces. Patient entered the hospital three days ago for examination in very much the same condition in which you see him tonight. We have, as you see, a complete paralysis of the right leg, no movement whatsoever being obtained. On the left side every movement is possible but is very weak. We find especially a weakness of the abductors and adductors of the leg; rotation of the left leg is almost impossible against the slightest resistance. A very interesting condition obtains in the muscles of the abdomen, as the abdominal wall below the naval is absolutely paralyzed, while the muscles above the umbilicus contract normally. If the patient tries to sit up without helping himself with the hands, the lower part of the abdomen protrudes, while the naval is drawn upwards towards the sternum. Examination of the sensory symptoms does not reveal any constant changes. There is a slight hyperesthesia, and slight hypoesthesia over both legs and a relative thermo-analgesia over the left leg, while the sense of heat and cold are normal in the right leg. There is a zone of hyperesthesia especially on the right side above the zone of paralysis. Examination of the reflexes shows that we have on both sides distinct Babinski, distinct Oppenheim, no ankle clonus; Achilles and kneejerks are present but feeble. The plantar reflex is present as well as the cremasteric, while the lower abdominal reflex is constantly absent. The upper abdominal reflex is mostly absent but can be obtained occasionally. Rectal examination shows that the sphincter contracts around the finger; otherwise normal condition. In the upper part of the body we find absolutely no symptoms and no eye symptoms. In the urine there is a good deal of pus found, and small amount of albumin corresponding to the amount of pus. Patient has had during the first three or four days temperature ranging up to about 103°, leukocytes of about 15,000. Fever has disappeared since yesterday and with it began a marked improvement in the condition, so that the motility of the left leg today is very much better than it was yesterday. The diagnosis of this condition is in the distinction between